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*TASTE AND THE ROLE OF EXPERIENCE
IN THE REGULATION OF FOOD INTAKE*

By HARRY L. JACOBS
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*with a Bibliography on the Sense of Taste prepared by
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14: Taste and the Role of Experience in the Regulation of Food Intake

by Harry L. Jacobs*

THIS PAPER will outline the problem of experiential factors in food intake, using the "sweet tooth" as a major example. We will focus on the following aspects of the problem. All meals contain nutrient and taste cues. What is the relationship between taste and nutrient properties of food in determining food acceptance? Is it learned? Is it innate? Is it both?

The term "taste" will be used to include all of the sensory complex usually subsumed under the rubric "flavor," including taste, smell, texture, viscosity, stickiness, etc. The discussion will be limited to laboratory studies on the rat and to the work of physiologists and psychologists interested in food intake.

Investigators in physiology and nutrition interested in the maintenance of energy balance have analyzed food intake in terms of neurophysiological and biochemical mechanisms. This approach usually assumes that organisms eat when they need food and that the relation between the need for nutrients and the acceptability of foodstuffs is innate and relatively independent of past experience.

Psychologists working on learning theory have also been interested in food intake, but from a different point of view. Their interest in hunger has been in the energizing properties of behavior. Hun-

ger drive makes the organism highly active; once behavior is started, all changes in its direction are investigated as problems in trial-and-error learning. Thus, in the case of food intake, the search for and ingestion of foodstuffs is assumed to be learned. All food is presumed originally neutral in incentive value; the relation between need for nutrients and the acceptability of foodstuffs is learned and is fully dependent on past experiences.

We will not refer to the literature on human subjects in this paper, nor will we review the general work on incentive motivation in animals. Reference to the latter will be limited to selected cases where the interest has been in consummatory responses rather than in instrumental behavior.

TASTE AND INTAKE— THREE INTERPRETATIONS

There are at least five items present in any particular instance of feeding behavior. The *ingesta* contains *taste* and *nutrients* which can act as potential modulators of *intake*. When organic needs or *deficits* are present, the ingestion of nutrients acts to correct them. Psychologists have referred to this relationship as need reduction.

Figure 14-1 presents three views of

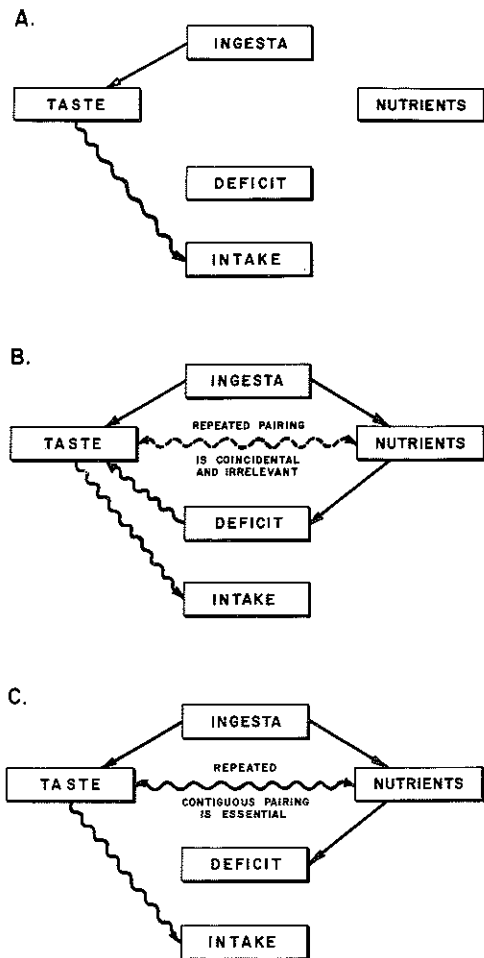


Fig. 14-1. Three interpretations of the role of taste in the control of food intake. A. Innate Incentives; acceptability as a function of chemoreceptor stimulation. B. Behavioral Regulation; acceptability as a function of innate needs and homeostatic mechanisms. C. Learning; acceptability as a function of the association of taste with organic need reduction.

the relationship between these five aspects of feeding. Inspection of the left side of each schema shows that they are similar in assuming that all ingesta produce taste cues (solid lines), and that taste acts to modulate further intake (wiggly lines). These diagrams differ in the role assigned to the necessity for occurrence of a deficit and its reduction by ingested nutrients.

The first approach, in the upper part

of Fig. 14-1, assumes that *innate* incentives regulate intake, irrespective of the nutrient content of a diet or the occurrence of a deficit. Young uses the term "palatability" to describe this approach, limiting it to cases where food acceptability is determined by stimulation of head receptors by characteristics of the food itself [29]. Troland postulates two general types of receptors, "beneceptors," and "nociceptors," which, when stimulated, give rise to pleasantness and unpleasantness, respectively [26]. The major evidence for this approach comes from observations of preference for specific foodstuffs in satiated animals, where nutrient values of the food choices are identical and the choice is apparently independent of past experience. For example, Scott and Verney gave rats a choice between diets containing various carbohydrates in granulated form. Since the nutritive value of the choices was considered equal, these authors concluded that "it is impossible, therefore, that selection . . . was based on any nutritional qualities of the choices, and probable that the appetites shown for these substances were trivial in origin (a function of innate incentives, in our terms)" ([21], p. 406).

The second approach to the role of taste in food intake, in the center of Fig. 14-1, is labeled *behavioral regulation*. It is similar to the first in assuming that the role of taste is innate, but it extends the assumption by asserting that the occurrence of a deficit is the critical factor because it potentiates taste in modulating intake (wiggly line between deficit and taste). The fact that nutrients are present and relieve the deficit is considered irrelevant (wiggly dashed line). The best example of evidence for this approach comes from observations on the specific appetite for salt in adrenalectomized rats (see Fregly paper, this symposium). As Richter points out in

his classic analysis of this case, "The results indicate that the adrenalectomized rats ingest salt, not because they learn that salt relieves their deficiency discomfort, but because of chemical changes in the taste mechanisms in the oral cavity, giving rise to enhanced salt discrimination" ([18], p. 370).

Richter may well be wrong about the specific sensory mechanisms mediating this shift in preference. The critical point is that the choice itself is mediated by innate physiological mechanisms. As Katz points out in his "avidity" theory of specific hungers, which assumes that need alters perceptual bias so that the animal seeks out and ingests the needed food, that learning may well be necessary in acquiring the instrumental responses involved in the *finding* of food [12]. However, the actual *choice* is determined by the increased acceptability of a particular set of taste qualities. As Fig. 14-1 points out, the fact that nutrients are also present is coincidental. The animal could be fooled by a substitute food identical in taste but containing no nutrients.

The third approach to the role of taste in food intake, at the bottom of Fig. 14-1, is labeled *learning*. In this view, both nutrients and the occurrence and relief of a deficit are critical. This approach assumes that taste cues are initially neutral stimuli. They gradually acquire the ability to modulate intake by conditioning, through repeated association of the taste of food with the deficit relieving effect of the nutrient. The first use of this approach in animal studies was in the classic work of Harris *et al.*, who described it in subjective terms: "...It depends not on a vague instinct but on an association between the distinctive character of the diet (smell, taste, appearance) and an experience of the prompt beneficial effects..." ([6], p. 187).

Hull [7], attacking the same problem in the context of modern learning the-

ory, is more objective, preferring to speak of effector responses (e.g., eating) as being conditioned to specific afferent discharges (in this instance, taste) contiguous with tissue need reduction (deficit relief). This approach has the advantage of allowing reference to a large body of independently derived principles available in learning theory. Thus, more precise predictions can be made in designing and carrying out experiments to evaluate this approach.

The rest of this paper will outline several studies designed to evaluate the role of taste in food intake from these three viewpoints.

THE ONTOGENY OF SACCHARINE PREFERENCE

Why does an adult rat have an appetite for saccharine? Although saccharine may have some physiological effects [27], it is non-nutritive. If this is so, saccharine may be an innate incentive (Fig. 14-1, *A*). In agreement with this possibility, Nachman [13] has shown that it is possible to breed rats with appetites and aversions for saccharine. However, the specificity of the genetic effect is still an open question, e.g., Nachman may merely be breeding for the ability to *acquire* saccharine preference.

The conditions of nurturance in the newborn rat are ideal for the application of the learning model (Fig. 14-1, *C*) to the ontogeny of saccharine preference. The argument, for any mammal, would run somewhat as follows. "Since taste buds in the babies' mouth are stimulated by milk sugar a little before the milk reaches the stomach, the argument that our liking for sweets is acquired by conditioning has some plausibility..." ([28], p. 684).

Since the neonate rat is completely dependent on its mother for food and presumably eats when hungry, the mild sweetness of mother's milk provided by

its 2.8 per cent lactose content [14] would have ample opportunity for contiguous pairing with deficit relief following each meal. Thus, the originally neutral sweetness of milk should become a conditioned incentive, establishing a stimulus generalization gradient so that sweet substances of greater or less concentration than the original conditioned stimulus should also be acceptable, though less so than the starting point, a 2.8 per cent lactose solution.

This argument generated some simple predictions. Neonate rats should show a gradual increase in appetite for 2.8 per cent lactose solution, and this learning curve of acquired preference value should be steeper than a very sweet 0.1 per cent saccharine solution, known to be palatable to the adult rat.

In order to test these predictions, we developed a reliable method for measuring acceptability of single drops of solution presented to neonate rats [10]. All subjects were raised with their mother and were removed for testing four times per day, at four-hour intervals. Figure

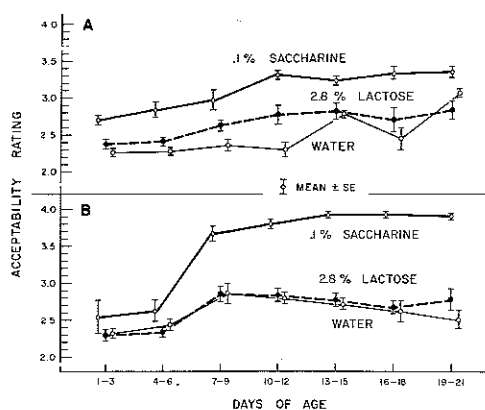


Fig. 14-2. Prewaning acceptability of lactose and saccharine solutions and water. A. Independent litters containing five pups each. B. Single four-pup litter tested on all solutions [10].

14-2 shows the results of our first series of experiments on this problem. In both

series of tests, the sweet 0.1 per cent saccharine showed increasing acceptability during the weaning period, while the 2.8 per cent lactose solution, which should have been more acceptable on the basis of our learning model, was not differentiable from water. In other experiments we increased the lactose percentage to 4 per cent (discriminable by adult rats) without effect. The only way we could make lactose palatable to the neonate rat was to make it sweeter. A 17 per cent lactose solution, far sweeter than the presumed original point of conditioning, finally produced an acceptability curve equal to the 0.1 per cent saccharine solution.

The acceptability curve for lactose was directly contrary to the predictions from the learning model outlined above. What about the increased intake of saccharine? Could this be learning, even though the reinforcement was unknown? Control experiments showed that this was not the case. Rats tested with 0.1 per cent saccharine for the first time at fifteen days of age required only one day to reach the asymptote of acceptability. We have taken this to indicate that the saccharine curve in Fig. 14-2 is due to the maturation of the indicator responses used in our tests, sucking and reaching out for a drop of solution, licking lips and face after swallowing, etc.

The above studies allow us to reject a simple learning model in interpreting the ontogeny of sweet preferences in neonate rats. We can now assume that saccharine preference in adult rats is not simply a case of stimulus generalization from early lactose conditioning.

SWEET PREFERENCE IN ADULT RATS

The problem of the relationship between deficit and taste still raises the question of learning in adult animals.

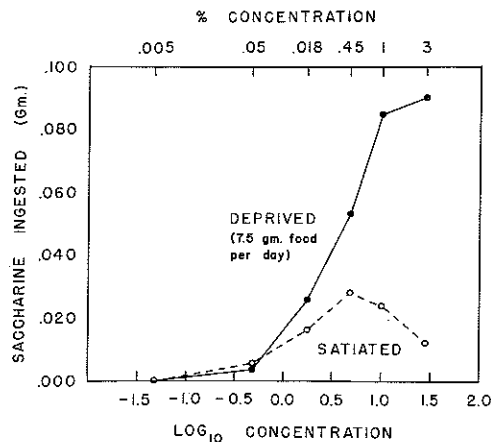


Fig. 14-3. The relative preference for sodium saccharine as a function of concentration in food-deprived ($N = 18$) or satiated ($N = 18$) rats. (Modified from [1].)

Figure 14-3 shows the results of a study by Bacon *et al.* [1] of relative saccharine preference in adult rats and its relation to a rather severe energy deficit. Using daily thirty-minute tests with single bottles, satiated rats show maximum preference for a mildly sweet 0.45 per cent solution. Food deprivation increases maximum preference to an extremely sweet, 3 per cent solution. These results are analogous to the kind of acceptability shifts demonstrable in specific hungers and raise the same questions.

Is it possible that energy deficit potentiates taste in regulating food intake in general, in the same way that adrenalectomy potentiates taste in salt preference? If so, does this operate on the basis of behavioral regulation, as Richter assumed in the case of specific hungers [20], or does the adult animal learn to like the taste because of deficit relief? Figure 14-3 suggests that the former may indeed apply, for hunger is potentiating the taste of saccharine (which lacks nutrients). What would happen if nutrients were added to the solution, i.e., if sugar were compared to saccharine? Le

Magnen [15] attacked this question directly by comparing the effects of hunger on sucrose (taste and nutrients) with its effect on saccharine (taste). Figure 14-4

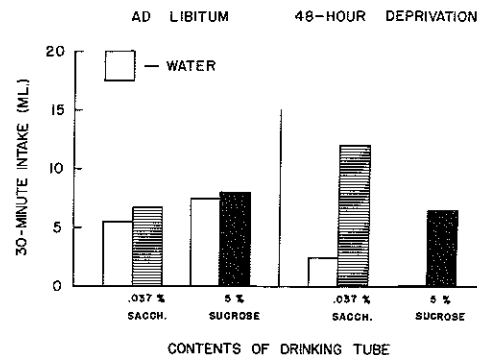


Fig. 14-4. The effect of food deprivation on the relative preference of sucrose or saccharine over water in two-choice tests. (Modified from [15].)

shows that food deprivation increases the intake of both solutions but that the relative change in preference is even greater in the case of saccharine. These results are in agreement with Richter's [20] and Katz's [12] assumption that deficit potentiates taste regardless of nutrients or deficit relief.

However, these are very short-term tests. What happens to the saccharine effect if the tests are repeated daily? How long will the animal be fooled?

Sheffield and Roby [22] carried out a series of eighteen daily tests in which hungry rats were given brief drinking trials in which saccharine was available. Figure 14-5 shows the results of this study. Deprivation continues to increase intake throughout the testing period. The authors use these results to argue against a learning interpretation of the data. If the drinking response had been learned in the first place, it would have shown signs of extinction during the test series.

Smith and Capretta [23] disagreed with the conclusions of this study, arguing that optimal conditions for produc-

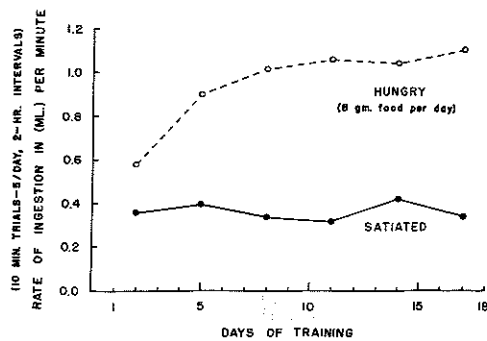


Fig. 14-5. The effect of food deprivation on the acquisition of drinking of 0.13 per cent saccharine solution in response to a specific cue-pattern accompanying availability of the solution. (Modified from [22].)

ing extinction had not been met. They designed a series of studies with rats adapted to a twenty-one-hour food deprivation schedule, precisely varying the opportunity for the animals to associate the ingestion of saccharine with contiguous nutrient ingestion and assimilation. The findings were quite consistent, showing that hungry rats did not continue to be fooled by saccharine if the drinking test was clearly and consistently separated from their daily meal [3,4,23].

Thus, as in Fig. 14-6, hungry rats that

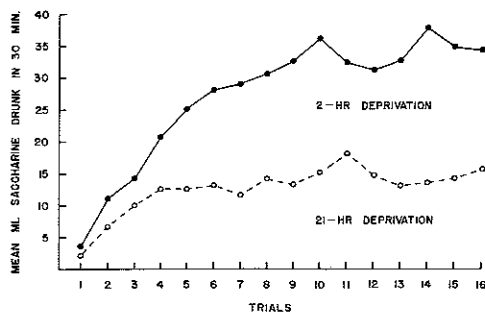


Fig. 14-6. The effect of hunger at the time of testing on saccharine intake. (N = 8 in each group.) Upper group, twenty-one-hour food deprived, fed two hours before test; lower groups, twenty-one-hour food and water deprived, fed thirty minutes after test. (Modified from [4].)

were fed two hours before the saccharine test (*top curve*) continued to ingest large

amounts of solution, presumably because of the continued opportunity to associate its taste with deficit relief. Hungry animals denied this opportunity ingested less saccharine (*lower curve*). Capretta interprets the latter case as extinction.

However, Capretta does recognize an alternative interpretation, i.e., that the animals that are fed while hungry may be developing a learned avoidance for saccharine because of its association with this unpleasant state of affairs. He tried to test this in the experiment shown in Fig. 14-6 by using hungry and thirsty animals (*lower curve*). He argues that adding water deficit to food deficit should increase unpleasantness. Thus, if this were a learned avoidance, these animals would show less intake (more avoidance) than the groups in his previous studies, who only had hunger to contend with. He reports that this did not occur and thus he rejects the learned-avoidance hypothesis.

Although Capretta's argument is reasonable, it may be premature. First, due to the well-known correlation between food and water intake [11], the addition of thirst to hunger may not increase total deficit as much as he assumed. Further studies, adding other avoidance drives, e.g., shock, to food deprivation, are needed to answer the question he raises. Second, it is possible to show learned avoidance for saccharine in rats. Garcia and his colleagues have repeatedly demonstrated a learned avoidance after drinking saccharine in the presence of low-level gamma radiation (e.g., [5]). This is clearly shown in Fig. 14-7, where twenty-four-hour tests of saccharine solution and water intake produce a relative avoidance of saccharine for several weeks.

In summary, more work is needed to clearly evaluate the extent to which the learning model applies to the changes in saccharine preference induced by food deprivation in adult rats.

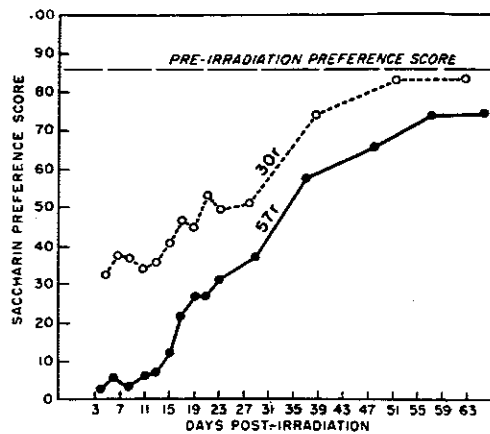


Fig. 14-7. The effect of drinking 0.1 per cent solution of saccharine during six hours of either 30r (upper curve) or 57r (lower curve) gamma radiation, on subsequent avoidance and recovery in a series of daily, two-choice tests between 0.1 per cent saccharine and water. (Modified from [5].)

LONG-TERM EFFECTS AND A TWO-FACTOR SYSTEM

The studies of food deprivation reviewed above were limited to intake tests of less than an hour's duration; long-term effects were measured by repeated daily measurements. What of the effects of deprivation on longer intake tests? I have studied this using insulin injections to induce hunger.

It has long been known that insulin hypoglycemia increases the intake of dry-stock diet or of glucose solutions (e.g., [19]). Soulaire [25] extended this technique by offering single-bottle choices of 10 per cent sucrose, glucose, or maltose. He found that the increase in intake was in direct proportion to sweetness. We verified this result using a choice test [8]. We found that insulin increased the intake of the sweeter solution over a three-day test period. At this point we became interested in the following question, quite like those presented above in our discussion of saccharine. Does this shift in appetite occur because the animal learns to like the sweeter solution, since it provides the

greater caloric density per unit of intake, or because of behavioral regulation making the sweeter solution taste better regardless of its caloric content?

We decided to attempt to answer this question by giving the rat a direct choice between a mildly sweet solution highly capable of relieving the deficit, and a much sweeter solution, less capable of relieving the deficit. Since insulin hypoglycemia produces a specific deficit for glucose [24], we chose this as our basal sugar. Our other choice was fructose, which is sweeter than glucose and much less efficient in relieving symptoms of hypoglycemia [2].

Rats were given two days of choice between water and 30 per cent solutions of glucose or fructose. Subcutaneous injections of insulin (PZI) or physiological saline were administered at twelve-hour intervals, at which time intake was measured. The insulin group received 2.0 units on the first day and 3.0 units on the second day. Table 14-1 shows the

TABLE 14-1: Number of Subjects Preferring Each Sugar Solution^a

Group	Insulin		Control	
	Glucose	Fructose	Glucose	Fructose
First day	1	10	6	5
Second Day	8	3	9	2

^a Preference defined in terms of relative total volume consumed during the 24-hour interval.

frequency of subjects preferring either solution during the two-day test. On day 1, insulin produced a significantly greater number of animals preferring the fructose solution ($p = 0.0119$). The insulin effect disappeared by the second day, on which both groups preferred the glucose solution. Figure 14-8 shows the average intake scores during the two-day test. Again, insulin produced a significant fructose preference for day 1 totals, which was sharply reversed in that significant preferences for glucose were

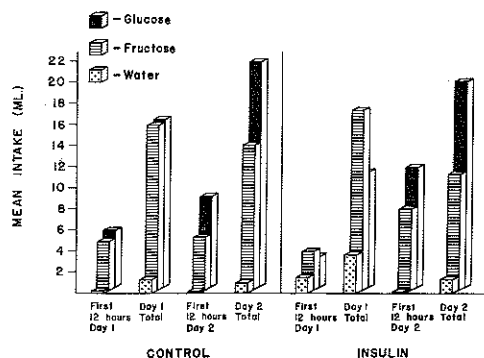


Fig. 14-8. The effect of insulin injections on the relative intake of 30 per cent solutions of glucose and fructose and water in a three-choice intake test. (N = 11 in each group.)

demonstrated during the first twelve hours on through the totals for day 2. The control group failed to show a preference for fructose during the two-day test. The glucose preference was clear but had not reached significance by the end of day 2.

In summary, insulin produced a significant preference for the sweeter fructose solution, apparently regardless of its inefficiency in relieving the insulin-induced deficit. If our observations were limited to day 1, we would conclude that behavioral regulation was potentiating sweet tastes, as in the saccharine cases described above. During the second day, however, the insulin-treated rats reversed their preference, now choosing the less sweet solution, which was very helpful in relieving the deficit. If our observations were limited to the second day, we would conclude that our learning model applied—the rats associating the taste of glucose with the deficit relief that followed its ingestion.

This is a very confusing situation. Apparently the rats took what they liked, presumably on an innate basis, on day 1, and took what they needed, presumably on a learned basis, on day 2. Although this explanation seemed quite *ad hoc* and overcomplicated to us, it would seem that it is not completely unreasonable.

Figure 14-9 shows a study by Le Magnen [15] of sucrose and saccharine intake in rats on five days of total food

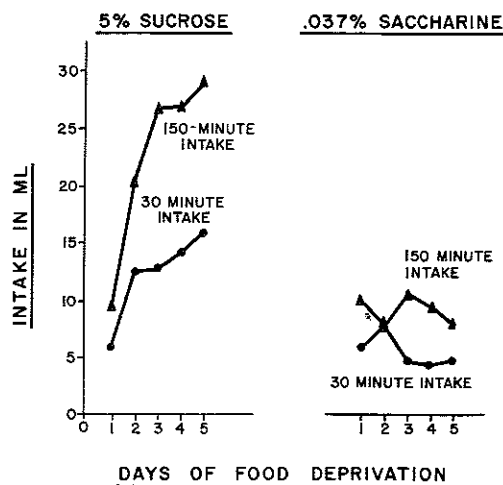


Fig. 14-9. The effect of food deprivation on the relative intake of sucrose and saccharine in daily three-hour single-bottle tests. (Modified from [15].)

deprivation. The rats were given a daily three-hour test. The intake was measured at "short-term," thirty minutes, and at "long-term," three hours. For the first day the thirty-minute intake showed saccharine to be higher than sucrose as in the work discussed in Fig. 14-4 above. This would be in agreement with a behavioral regulation view, as in the first day of our insulin studies. Following Fig. 14-9 again, the intake of sucrose kept increasing over the five-day deprivation period, while that of saccharine was variable and even tended to decrease. The latter result would be in agreement with a learning interpretation of the data, as in the case of the second day of our insulin studies.

I would like to paraphrase Le Magnen's interpretation of this type of study [15,17]. He distinguishes between two distinct and successive mechanisms regulating food acceptance. The *primary response* acts on a short-term basis, and the animal responds immediately by using flavor or other sensory qualities as a

cue for ingestion, regardless of whether it corrects the nutritional disequilibrium. As the animal is allowed a series of trials lasting long enough for the ingesta to be assimilated, a *secondary response* develops, which is no longer specific to the sensory qualities of the food, but depends upon its capacity to repair the nutritional disequilibrium. In summary, Le Magnen's interpretation of his data is made in terms of a two-factor system combining the properties of what we have called behavioral regulation and learning in Fig. 14-1 above.

LEARNING AND THE ROLE OF SENSORY STIMULI IN SATIETY

Most of us tend to analyze the problem of satiety by assuming that nutrients supply the cues for the cessation of eating [9]. Thus, theories of food intake have really been theories of satiety, in which a particular metabolic change carries the burden of explanation, e.g., the "glucostatic" theory, "thermostatic" theory, "lipostatic" theory, and in recent discussions, an "aminostatic" theory. Le Magnen [17], in a thorough analysis of the role of sensory qualities (taste, in our terms) in the control of food intake, points out that metabolic cues are not

very useful in precise control of adult food intake. He argues that animals learn to make use of sensory cues and that these cues can be equal to or even more important than metabolic cues in controlling rate of intake.

I shall briefly outline two examples of his work along these lines. In both cases he varied the sensory qualities of the diet by adding olfactory cues. First, he considered the case of satiety induced by associating the diet with insulin-induced hunger [16]. Rats were given a thirty-minute meal of a diet flavored with either citral or eucalyptal. Six intake tests were run each week, each rat alternating between diets. Two groups of rats were tested, each over a twenty-day period. In one group the citral was followed by injections of insulin; in the other group, the eucalyptal diet was followed by insulin. After twenty days of testing were completed, both groups were given a free choice between the two diets without insulin injections. Figure 14-10 shows the results of this study. During the twenty-day test period, the intake of the diet that was followed by insulin injections showed a sharp decrease. Unlike the ambiguous changes in Capretta's experiments discussed above, this is almost certainly another example of a

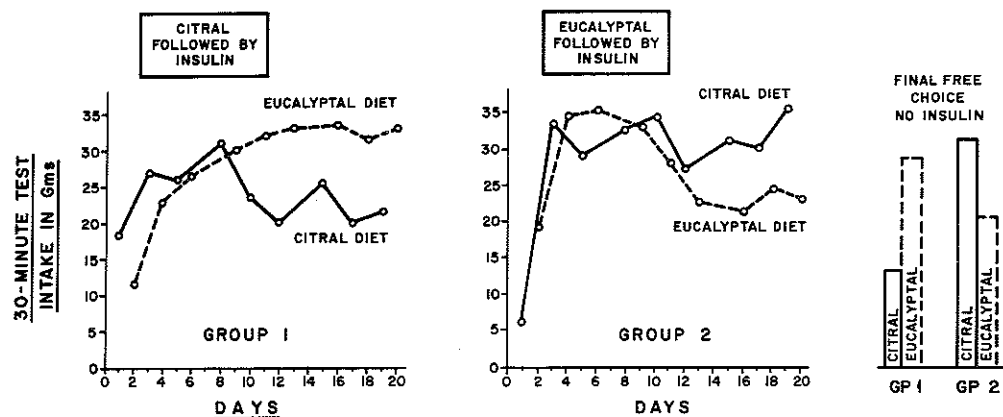


Fig. 14-10. The effect of subcutaneous insulin injections (0.05 U/kg), following twenty daily meals containing a specific olfactory cue pattern, on its subsequent intake in a free-choice test. (Modified from [16].)

clear-cut conditioned aversion, which carries over to the free-choice test, where the intake of the diet previously followed by insulin was depressed in proportion to the insulin effect.

The same kind of change can be shown by allowing the animal to associate an olfactory stimulus with satiety signals from an intragastric load of glucose [17]. Rats were given two one-hour meals per day, each with a discriminable olfactory cue added, as in the experiment just discussed. A constant pattern of feeding was established in which one meal was followed by a glucose load, the other by saline. Figure 14-11 shows the

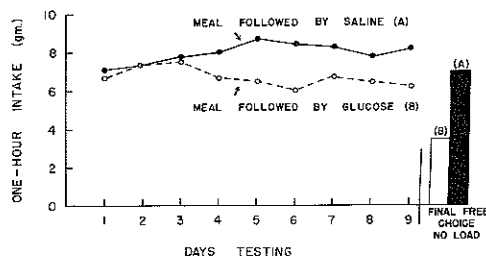


Fig. 14-11. The effect of intragastric glucose loads, following nine daily one-hour meals containing a specific olfactory cue pattern, on its subsequent intake in a free-choice test. (Modified from [17].)

results of this study. The meal followed by glucose showed a decreased intake, thereby reflecting the satiety value of the intragastric load. The final free choice again showed that conditioned satiety can be demonstrated. Le Magnen reports that the same effect can be obtained with D-amphetamine to induce satiety [17].

In summary, both of these examples clearly show the ease with which metabolic signals (changes in blood sugar level in these cases) can use sensory cues (via conditioning) as a vehicle to regulate intake. Le Magnen speculates that this is perhaps a dominant way of controlling intake in adult, "sophisticated" animals living in an environment where many food choices are available.

SUMMARY

This paper has outlined the problem of the roles of taste and nutrients as cues controlling food intake with special emphasis being placed on experimental factors. Studies of saccharine and sugar preference, or the appetite for flavored diets, in neonate and adult animals were reviewed. The effect of deficits induced by food deprivation or insulin injections in these studies was evaluated, leading to the over-all conclusion that deficits potentiate taste in the control of intake, and that these changes have both innate and learned components, the former on a short-term basis, the latter on long-term basis.

Discussion

Grossman: You are assuming temporal contiguity between taste and the nutritive effects of the ingested foods. Such contiguity certainly does not exist, and the delay of reinforcement is much greater than anything shown to be effective in any laboratory situation. How can you assume that learning or conditioning will take place under those conditions?

If this type of an association is, in fact, possible, I fail to see how your saccharine data rule out the possibility that the saccharine preference may be learned through an association of the sweet taste with nutrient effects of other sweet substances.

Jacobs: It is quite true that contiguity is seldom present. In Hull's famous footnote on the status of food as a reinforcer, he assumes that the temporal gap between mastication and need reduction is filled by a chain of secondarily reinforced associations, and that primary reinforcement may not operate at all (Hull, *Principles of Behavior*, Appleton-Century, New York, p. 98, 1943). Hull's analysis of this problem was developed

in a rather artificial laboratory situation, of course.

As to the second question, I purposely chose the natural situation as one in which maximum opportunity was allowed for the association of the sweet taste with nutrient effects. If this association did take place, the maximum point of reinforcement should have been the 2.8 per cent lactose solution contained in rats' milk. The sweeter saccharine solution should have been farther out on the gradient of secondary reinforcement. Thus, it should have been less palatable. This did not happen, a fact which is not in agreement with the learning hypothesis.

I am not insisting that this is the only approach to the problem of learning. I once tried some experiments with chicks, depriving them of the opportunity to taste sweets from birth by putting them on a carbohydrate-free diet. As adults, the sweetness "deprived" chicks preferred sucrose as much as a group maintained on a high sucrose diet. Thus, early experience made no difference, which is also against the learning hypothesis.

Epstein: The interesting experiments of Le Magnen could be interpreted in the following way. After the treatment with insulin and glucose, the rat in a subsequent choice situation chooses the diet in which he has experienced the least anorexia. This is confirmed by the fact that Le Magnen has also used amphetamine with essentially similar results. Moreover, all of these differential appetites disappear if the choice is made after overnight deprivation. The animal becomes less discriminating.

Jacobs: I have seen secondary sources on this point; if this is a reliable effect, your interpretation may well apply.

Pfaffmann: Garcia (*Science*, 122: 157,

1955) essentially reproduced what was demonstrated by M. Nachman (*Amer. J. Physiol.*, 205 [2]: 219-21, 1963) in my lab, i.e., the effect of long, slow poisoning or radiation, acting slowly to make the animal feel sick, which is attached to the taste of a particular food. That same treatment is specific to the spot where the poisoning took place. There appears to be a differential connection between tastes of foods and poisoning effects and the place where the poisoning has occurred. It is not just a homogeneous situation. There is a relevance between the stomach changes and the taste which produces that which obviates the delayed reinforcement problem. You cannot get the animal to show bait shyness for the place where the poisoning is taking place. This seems to follow the reinforcement difficulty. There is a whole area here which has not been investigated from a naturalistic point of view and which may point out that learning conceptions are, in fact, arbitrary.

Jacobs: I agree. Psychologists have been very proficient in designing simple, artificial techniques that give standard measures of learning and precise, reliable, and occasionally irrelevant results, at least in complicated situations such as these.

De Ruiter: With respect to Grossman's comment that there may be too long a delay for learning to be possible from the effects of ingestion, this may not be so in the case of sugars. We find that when a fasting rat starts a meal, its blood glucose concentration rises to saturated levels within some fifteen minutes. This is surprisingly rapid, and we are not sure what is behind it yet, but the delay may not be so very long.

Kare: Jacobs implied that "sweetness" is of consequence to chicks. We have measured the response to sugar solutions of

thousands of birds, including chickens, gulls, blackbirds, starlings, robins, quail, and many others. We have yet to observe a preference for sucrose solutions when such birds are on an adequate diet (Kare, in Sturkie, *Avian Physiology*, Cornell University Press, Ithaca, 1965).

A commonly repeated suggestion that animals share man's "sweet tooth" receives limited support from experimental evidence. The failure to respond to so-called sweet solutions by many members of the class *Aves* is not unique. For example, the cat (Frings, *Experientia*, 7: 424, 1951), the armadillo (Maller and Kare, *Anim. Behav.*, 1967 [in press]), and some species of fish do not display an avidity for sugars. On the basis of results available, one could similarly argue for the universal appeal of short-chain fatty acids. A generalization on the response to sweetness in animals would be premature since this sense has been studied in only a few of the millions of species.

An appraisal of the evidence on sugar preference among animals reveals that the specific sugar under study is often undefined. Consideration of specific sugars further weakens an argument for the universality of sweet perception. For example, maltose is one of the sugars that rats prefer most, but man derives little or no sweet sensation from it. The calf, which responds to sucrose solutions even at concentrations insipid to man, is indifferent to maltose while the armadillo is offended by this sugar. Obviously, the criterion of sweetness, as defined by man for specific sugars, does not freely transcend species boundaries.

A limited effort has been made in taste studies to ascertain if the response to a sugar is based upon nutritive value. The polysaccharides, starch and glycogen, which do not share the common sugar's sweetness, have not been compared in regard to acceptability. In fact, in the

literature, equimolar solutions rather than isocaloric solutions are often compared, confounding these two parameters. We have demonstrated that the nutritive state will modify preference behavior for sucrose; therefore, it would be desirable in a taste study to divorce taste from nutritive value. The use of synthetic sweeteners—saccharine, dulcin, and sodium cyclamate—provides this opportunity. Unfortunately, however, the majority of animals tested with these chemicals are indifferent to or offended by their taste.

In our preference studies on sugar we have searched for physiological, chemical, or physical variables of sugar solutions that might be associated with their selection. We considered circulating blood glucose level (Kare and Ficken, in *Olfaction and Taste*, Y. Zotterman [ed.], Pergamon Press, New York, 1963) and milk sugar level. However, no physiological variable can be offered to explain the collective comparative results. We have examined preference data in terms of osmotic pressure, viscosity, melting point, configuration, and conformation, but could find no common denominator that might explain the difference in taste preference of sugars between species.

A second but minor point is that your slides seem to show a comparison between the responses to sugar and saccharine. Are these substances supposed to be near equal in sweetness? If we use the rule of thumb as applied to man, that saccharine is approximately 200 to 400 times as sweet as sucrose, there is a substantial difference in taste intensity between your choices.

Jacobs: That is a pretty broad rule. As to your point on whether chicks discriminate "sweetness," I agree that these data are unclear (Kare and Halpern [eds.], *The Physiological and Behavioral As-*

pects of Taste, University of Chicago Press, Chicago, 1961, p. 30.)

Kare: In the case of Le Magnen's data, he used 5 per cent sucrose and saccharin equivalents of 3.7 or 7.4 per cent, but yours were all the way up to 26–52 per cent. At these two levels, I do not think saccharin can be freely compared with the sugar solutions. I don't know of any animal that responds to 2.8 per cent lactose. I don't know how to interpret your conjectures on learning, if you are using a stimulus that may be meaningless to the rat.

Jacobs: I agree that conjectures on absolute sweetness levels may be meaningless. In my case I merely had to assume that 0.1 per cent saccharin was sweeter for the rat than a 2.8 per cent lactose solution.

Kare: It is difficult to get a substantial response from the rat to 2.8 per cent lactose; the opossum is unique in that it responds very well to lactose.

Jacobs: The lactose problem is complicated, even in rats. For one thing, lactose is toxic to adult rats. Adult rats can discriminate lactose from water at 4 per cent concentration (Richter and Campbell, *J. Nutr.*, 20: 31, 1940). The learning hypothesis would have run into logical difficulty even if I had not carried out my experiments on neonates. If the neonate rat presumably learns to like sweets on the basis of mildly sweet 2.8 per cent lactose solution as a conditioned stimulus, why does the adult rat avoid *all* lactose solutions it can discriminate, while still liking low concentrations of other sweet substances? One would have to add all sorts of complicated *ad hoc* hypotheses about learned avoidance, gradients of secondary approach and avoidance, to explain this.

As to the discriminability of a 2.8 per cent lactose solution, it is possible that the neonate is more sensitive than the adult animal. In any event, I have also tried 4 per cent lactose in the neonate and it responds to it as to the 2.8 per cent solution. I had to increase sweetness to 17 per cent to change acceptability in my neonate subjects.

Kare: We can marshal examples to support the contention that animals will avoid harmful compounds, but the basis for avoidance is not necessarily taste. Further, the response to a toxic compound is unpredictable and is not uniform across species.

Under some circumstances, lactose is toxic. However, the fowl and the cat accept a 5 per cent solution indifferently. While the rat does not strongly prefer lactose solutions, it avidly selects galactose, the constituent monosaccharide to which is ascribed the lactose toxicity (Perry *et al.*, *Acta Paedia*, 45: 228, 1956). Lactose is not the only sugar for which harmful aspects have been recognized. Xylose is recognized to have a deleterious action on the visual apparatus. Despite this, man finds it sweet and pleasant, the cat is indifferent, while the fowl actively rejects it in a choice situation.

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